C42. Eisenmenger Syndrome, Could Its Pathophysiological Changes Serve as Protective Factors Against Severe COVID-19 Infection?

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Background: Congenital heart disease (CHD) and pulmonary artery hypertension (PAH) are comorbidities to severe COVID-19 infection. We reported a case of adult congenital heart disease (ACHD) with Eisenmenger Syndrome (ES) who survived COVID-19 without progression to severe infection.
Case Description: A 39 years old male with history of uncorrected ACHD complained of low-grade fever and cough for 3 days. Initial oxygen saturation was 85%, no tachypnoea, no tachycardia, and blood pressure was normal. SARS-CoV2 RT-PCR results was positive. Thorax CT showed lung fibrosis, minimal GGO, and prominent pulmonary notch. Notable laboratory result was haemoglobin: 20.4g/dL. Echocardiography showed a large secundum ASD with bidirectional shunt, dilated RA and RV, normal ventricular systolic functions, mild TR with increased tricuspid gradient (TR V max 4.27m/s) which concluded the diagnosis of ES. The patient was treated according to moderate COVID-19 guidelines and ES management. His response to treatment was favourable and was discharged in 3 weeks uneventfully.
Discussion: Studies have documented CHD and PAH as comorbid to severe COVID-19 infection, but our patient had moderate COVID-19 infection and recovered without progression to severe disease. We proposed several pathophysiological characteristics of ES which could serve as protective factors against severe COVID-19, which are: 1) Pre-existing PAH, where pulmonary vasculopathy and remodelling lessened endothelial response to inflammation, and reduced expression of ACE2 receptors, 2) Chronic hypoxia, which prepared cells and tissues against hypoxic environment, and 3) Polycythaemia, might be beneficial in haemoglobinopathy directly caused by SARS-CoV2.
Keywords: Eisenmenger syndrome • PAH • hypoxia • protective • COVID-19.